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Photorespiration : A possible index for drought resistance in plants

(rice/*Oryza sativa* L./drought resistance/photorespiration/water stress)

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ABSTRACT Effect of water stress on photorespiration was studied in two genotypes of rice (drought tolerant N-22, drought susceptible Jaya) to establish the relationship between drought tolerance and photorespiration. Photorespiration, as an index of drought resistance is suggested.

Plants differ markedly in their tolerance to water deficit stress. Plants subjected to water stress have alterations in physiological, biochemical and enzymatic levels[1-3]. Photorespiration is an important physiological process, but has been considered as a wasteful process which, if eliminated, could result in the benefit of higher yields in agriculturally important crops. The relationship between water potential and processes generating carbon dioxide have received less attention, particularly in intact plants. Genotypes have been reported to differ in their tolerance to water stress[4], and photorespiration has been shown to be inhibited by water stress[5].

The present study was planned to find out relationship between drought tolerance of genotypes and water stress. Rice was choosen as test crop because water is an important factor in realizing maximum yield in this crop.

Seeds of two rice genotypes, a drought tolerant (N-22) and a drought susceptible (Jaya) were grown in the plastic trays according to standard plant sand culture method. Twenty one days old seedlings were exposed to water stress by dipping the roots in the PEG-6000 solutions of different water potentials for 4 and 6 h under light. Photorespiration was measured according to Ghildiyal and Sinha[6]. Relative water content was measured according to Kramer[7].

The changes in relative water content and photorespiration recorded, are tabulated in Tables 1 and 2 respectively. Photorespiration has been shown to be inhibited by water stress[8]. Until now however, there is no report that we are aware of, provides any direct determination of photorespiratory activity at mild water stress. Our experimental results have shown that contrary to prevailing assumptions that photorespiratory carbon dioxide evolution increased at

mild water stress and decreased when stress became increasingly severe. This is also interesting to note that drought tolerant genotype N-22 has low photorespiratory rates than of susceptible genotype Jaya ;

TABLE 1

Effect of water stress on relative water content (%) in the leaves of two genotypes of rice

Water potential of PEG-6000 solutions (Bar)	Jaya		N-22	
	Duration of water Stress (Hours)			
	4	6	4	6
Water (0)	96 0	96.5	95.8	96.2
-3.0	92.5	89.7	92.1	89.3
-5.0	90.1	87.2	90.0	83.6
-7.5	87.3	83.1	87.2	83.6
-10.0	85.8	80.2	85.3	80.2

TABLE 2

Effect of water stress on photorespiration in the leaves of two genotypes of rice ($^{14}CO_2$ evolved 1×10^4 dpm cm^{-2} h^{-1})

Water Potential of PEG-6000 solutions (Bar)	Jaya		N-22	
	Duration of water Stress (Hours)			
	4	6	4	6
Water (0)	5.87	6.20	4.10	4.00
-3.0	8.64	9.52	4.51	4.62
-5.0	6.38	6.34	4.18	3.99
-7.5	4.89	4.49	4.10	3.79
-10.0	4.52	4.01	3.70	3.40

and also activation and inhibition of photorespiration at mild or severe water stress was more sensitive in drought susceptible genotype Jaya. The drought tolerant genotype N-22 is thus characterized by low photorespiratory activity.

The present study thus suggests that photorespiratory activity under normal growth conditions and during the water stress might provide a measure 'of drought tolerance in the different genotypes of crop plants and can be used as an index for drought resistance.

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Diurnal cycle of physico-chemical factors in Mukhra pond, Bhagalpur

(diurnal cycle/physico-chemical factors)

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ABSTRACT Diurnal fluctuations of certain physico-chemical properties were studied in Mukhra pond of Bhagalpur over a period of 24 hours during monsoon, winter and summer seasons. Marked diurnal variations have been observed in respect of most of the physico-chemical factors.

Less attention has been paid towards the diurnal study of lentic waterbodies of Indian subcontinent[1-5] in comparision to temperate region. In the present study an attempt has been made to describe the general pattern of changes that occurred during a 24 hour period on a seasonal basis.

Water samples from the surface, middle and about 25 cm above the bottom of the pond were collected four hourly for 24 hours from 10 a.m. on one day to 10 a.m. the next day on three days *viz.* 17th to 18th August, 1983 representing the monsoon season, 11th to 12th January 1984 representing the winter season and 9th to 10th May, 1984 representing the summer season. Physico-chemical parameters were estimated following the standard methods of APHA[6].

The results of vertical variations in the physico-chemical factors in different layers have been depicted in Tables 1, 2 and 3. Water temperature showed a definite diurnal trend of enhancement during day time and decrease dur ng night hours. No thermal stratification of permanent nature was observed. Temporary stratification was visualized at 2 p.m. in all the seasons. It was maximum on 9th May, 1984 when the temperature difference between the surface and bottom water was 4.5°C as compared to 3.5°C on 17th August, 1983, and 3.6°C on 11th January, 1984. Transparency showed an upward trend from 10 a.m. to 2 p.m. and then there was a declinig tendency. It might be due to change in the intensity of solar radiation and shallow nature of the waterbody.

Dissolved oxygen value increased during day hours which fall down either after 2 p.m. or after 6 p.m. Depletion in its value during night was due to utilization in respiratory activity of plants and animals present in the pond. Stratification in dissolved oxygen value was not pronounced in the present study. pH value increased during day and showed downward trend during night hours. Maximum difference between bottom and surface value of pH (0.6) was detected at 6 p.m. on 11th January 1984.

Marked fluctuation in total alkalinity values was observed. It increased considerably during day hours which may be attributed to increased washermen's activity adding different types of bicarbonates in the form of detergents.

TABLE 1

Diurnal variation in the water chemistry in August, 1983

Factors		17.8.1983				18.8.1983		
		10 a.m.	2. p.m.	6. p.m.	10. p m.	2 a.m.	6 a.m.	10 a.m.
Temperature (°C)	S	31.5	32.4	30.6	29.8	29.6	27.6	30.9
	M	28.6	29.1	27.9	27.7	27.5	27.2	28.1
	B	28.2	28.9	28.0	27.5	27.4	26.8	27.8
Transparancy (cm)		48.0	54.0	41.5	–	–	36.5	47.5
Dissolved oxygen	S	10 8	11.6	10.4	9.3	8.6	8.5	9.5
(mg/l)	M	10.6	11 2	10.1	9.2	8.9	8.7	10.4
	B	9.8	10.4	8.6	8.0	7.9	7.8	8.9
Hydro.en-ion-	S	8.1	8.6	8.5	8.3	8.2	8.2	8,5
concentration	M	8.3	8.4	8.4	8.2	8.1	8.3	8.3
	B	8.4	8.3	8.3	8.1	8.0	7.9	8.0
Total alkalinity	S	119.0	152.0	148.0	133.0	112.0	110.0	118.0
(mg/l)	M	108.0	123.0	120.0	118.0	106.0	108.0	112.0
	B	106.0	110.0	108.0	105.0	101.0	104.0	117.0

S Surface
M Middle
B Bottom

TABLE 2

Diurnal variations in the water chemistry in January, 1984

Factors		11.1.1984				12.1.1984		
		10 a.m.	2 p.m.	6 p.m.	10 p.m.	2 a.m.	6 a m.	10 a.m.
Temperature (°C)	S	24.5	28.3	24.0	23.8	23.2	23.0	24.1
	M	23.8	24.9	23.2	23.1	22.5	22.2	23.4
	B	23.5	24.7	23.2	22.9	22.0	23.0	23.6
Transparency (cm)		56.4	61.2	51.8	–	–	44.6	53.8
Dissolved oxygen	S	12.2	12.5	13.0	12 0	11.6	11.2	12.1
(mg/l)	M	10.8	11.5	11.1	10.1	9.5	9.0	10.6
	B	10.6	10.2	9.7	9.0	8 8	8.8	9.6
Hydrogen-ion-	S	8.1	8.3	8.4	8.0	7.9	7.8	8.0
concentration	M	8.0	8.1	8.0	7.8	7.6	7.6	7.9
	B	7.8	7.9	7.8	7.6	7.6	7.7	7.8
Total alkalinity	S	115.0	122.0	124.0	118.0	110.0	108.0	112.0
(mg/l)	M	107.0	110.0	108.0	98.0	94.0	101.0	108.0
	B	106.0	113.0	115.0	112.0	99.0	106.0	106.0

S Surface
M Middle
B Bottom

TABLE 3

Diurnal variations in the water chemistry in May, 1984

Factors		9.5.1984				10 5.1984		
		10 a.m.	2 p.m.	10 a.m.	6 p.m.	2 a.m.	6. a.m.	10 a.m.
Temperature (°C)	S	38.4	40.0	37.2	37.0	36.1	36.0	37.5
	M	35.0	36.4	36.1	35.9	35.4	35.8	36.1
	B	34 6	35 5	35.0	34.2	34.0	34.6	35.0
Transparency (cm)		51.0	54.8	42.5	–	–	42.0	48.9
Dissolved oxygen	S	5.3	6.8	5.9	5.5	4.1	4.6	5.8
(mg/l)	M	4.9	5.2	5.0	4.8	4.3	3 9	5.1
	B	4 2	4.7	4.0	3.8	3.4	4.0	4.3
Hydrogen-ion-concentration	S	7.5	7.7	7.6	7.5	7.3	7.2	7.2
	M	7.4	7.6	7.2	7.3	7.1	7.1	7.4
	B	7.2	7.4	7.5	7.4	7.3	7.4	7.5
Total alkalinity	S	162.0	171.0	158.0	155.0	145.0	148.0	160.0
(mg/l)	M	148.0	156.0	154.0	144.0	140.0	132.0	139.0
	B	141.0	152 0	153.0	146.0	138.0	135.0	143.0

S Surface
M Middle
B Bottom

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Cytological behaviour of a multiploid in pearl millet (*Pennisetum americanum* (L.) K. Schum)

(multiploid/*Pennisetum americanum*)

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ABSTRACT An account of cytological behaviour of multiploid in *Pennisetum americanum* is described.

In the progeny of an open pollinated interchange trisomic, a multiploid plant was isolated in which PMC's were found with variable chromosome numbers ranging from 15 to 120/cell (Table 1). Of these, about 45% of PMC's exhibited $6^{II} + 1^{III}$ and 28.6% of PMC's were observed with $7^{II} + 1^{I}$ at MI. In addition, a number of PMC's were seen with different multiples of 15 chromosomes (Table 1). The probable chromosome associations like 60^{II} (Fig. 1), 45^{II}, $4^{II} + 52^{I}$ (Fig. 2), $5^{III} + 10^{II} + 85^{I}$ (Fig. 3), $3^{III} + 18^{II} + 75^{I}$ (Fig. 4), $22^{II} + 16^{I}$ and $20^{II} + 5^{I}$/cell were found in 7.6%, 5.7%, 2.9%, 1.9%, 2.9%, 1.9% and 2.9% of PMC's, respectively. The fact that inspite of presence of many cells with different multiples of trisomic chromosome number (15), the multivalent association were lacking barring a few trivalents observed in rare PMC's (Figs. 3 and 4) of this plant. In most of the multiploid PMC's, the bivalent associations were predominant, and the occurrence of univalents alongwith bivalents were relatively less common (12.5%). At anaphase I, 8 : 7 distribution of chromosomes was found in 81.1% of PMC's and 5.6%, 2.2%, and 11.1% of PMC's were seen with 23 : 22, 65 : 55 and 60 : 60 distributions, respectively (Table 1).

In case of multiploid, it appears that most of the univalents have been derived from bivalents at later stages of meiosis. Concomitantly, the predominance of only bivalents and a complete lack of multivalent assossiation in many multiploid PMC's of this stock

TABLE 1

Chromosome associations at metaphase I and distribution at anaphase I in multiploid pearl millet

No. of cells analysed	$6^{II} + 1^{III}$	$7^{II} + 1^{I}$	60^{II}	45^{II}	$4^{II} + 52^{I}$	$5^{III} + 10^{II} + 85^{I}$	$3^{III} + 18^{II} + 75^{I}$	$22^{II} + 16^{I}$	$20^{II} + 5^{I}$	Anaphase I distribution				
										Total No. of cells	8-7	23-22	65-55	60-60
105	48	30	8	6	3	2	3	2	3	90	75	5	2	10
Mean per cent	45.7	23.6	7.6	5.7	2.9	1.9	2.9	1.9	2.9	Mean per cent	81.1	5.6	2.2	11.1

further suggest that probably certain gene mutation causing bivalentization has restricted the formation of multivalents. A case of monofactorial "multiploid sporocytes" condition in pearl millet has been reported by Pantulu and Manga[1].

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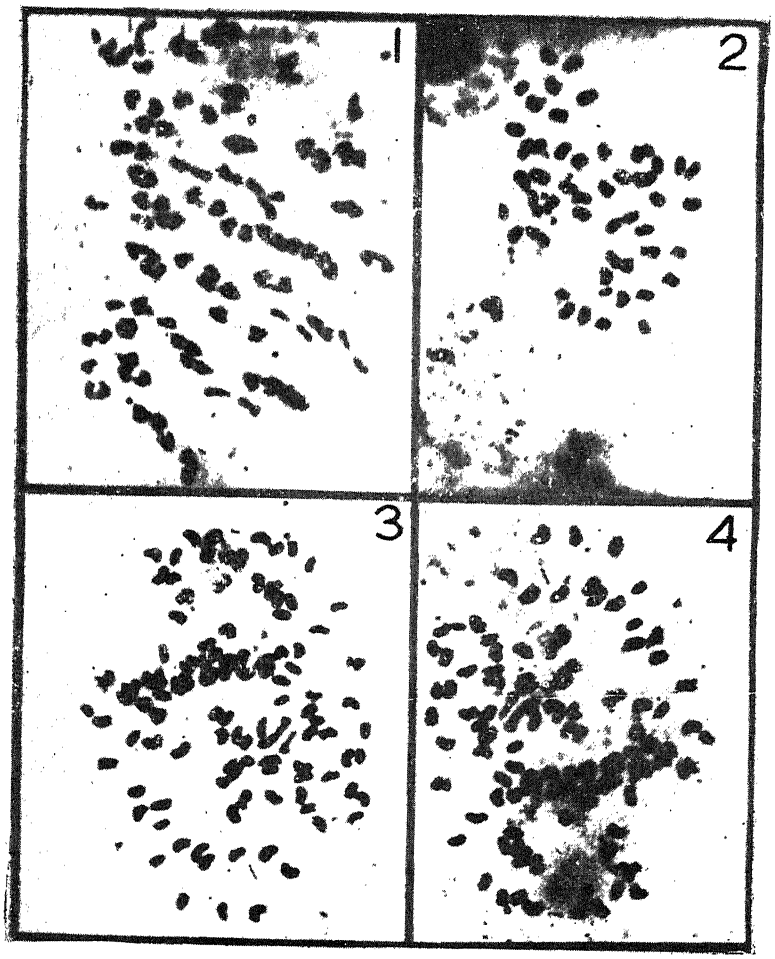
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Spectral properties of Ir(III) and Pt(IV) chelates of ketoanils

(I.R. spectra/absorption spectra/ketoanils/chelates)

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ABSTRACT Chelates of Ir(III) and Pt(IV) with 3-phenanthrylglyoxal-p-diethylaminoanil and 9-anthracylglyoxal-p-diethylamino-p-chloro, -p-bromo and -p-iodo anils have been synthesized and characterized by elemental analysis, and infrared, magnetic and electronic spectral measurements for their structures and bonding. All the ligands are coordinated with metal ions through azomethine and carbonyl groups in an octahedral fashion.

Coordination properties of less common noble metals including platinum metal in the environment of variety of organic ligands are well documented, however, their complexes with ketoanils which as ligands have interesting features[1-3] of forming isomeric and polymeric coordination species generally of unusual stereochemistries, have rare mention[4,5]. Scarce knowledge in the coordination chemistry of platinum metals complexed with ketoanils generated considerable interest to synthesize and characterize a few compounds of Ir(III) and Pt(IV) with 3-phenanthrylglyoxal-p-diethylaminoanil, and 9-anthracylglyoxal,-p-diethylamino,-p-chloro,-p-bromo, -p-iodo anils (abbreviated as PGDEA and AGDEA, AGCA, AGBA, AGIA respectively) using elemental analysis, infrared, magnetic and electronic spectral measurements in continuation of our previous work[6]. General structure of ligands is given bleow ;

$RCOCH{=}N\text{-}C_6H_4X$ - p, where R = phenanthryl or anthracyl nucleus and X = $N(C_2H_5)_2$, Cl, Br, or I.

Preparation of ligands and complexes ; All the five ketoanils[6] were precipitated either at room temperature or at ice temperature from the solutions in ether, containing both the reactants (glyoxal and amine) in equimolar quantities. Precipitates washed with ice cold ether were recrystallized from chloroform.

On mixing acetonic equimolar solutions of ligands and metal chlorides together in appropriate proportions, complexes were precipitated. Products washed with ether were recrystallized from methyl alcohol and were dried over anhydrous calcium chloride at low pressure. BDH or JM (London) laboratory reagents were used as supplied in the preparative work.

Analysis and physical measurements ; Nitrogen in the complexes was estimated at C. D. R. I., Lucknow. Platinum and Irridium were determined[7] as metals after decomposing the complexes with mixtures of H_2SO_4 and HNO_3 and HCl successively; excess acids were evaporated and the aqueous dilute solution was reduced with sodium acetate and formic acid. Precipitated metal was collected on Whatmann '42' filter paper and was washed with water to remove chloride ions. Dry filter paper was ignited to constant weight. Chlorine was estimated after decomposing the complexes with H_2SO_4 and HNO_3 mixture, gravimetrically as AgCl.

IR spectra were recorded in nujol mull medium on Beckmann DU-621 spectrophotometer in 200 to 4000 cm^{-1} range. Magnetic measurements were made on the pulverized samples by Gouy's method. Optical density observations in ultraviolet and visible regions were noted on the standard solutions prepared by dissolving known quantities of complexes in known volume of methyl alcohol with the help of Beckmann DU-2 spectrophotometer.

Analytical results of the complexes correspond to their molecular formulae given in Table 1.

TABLE 1

Electronic, spectral and magnetic data

Complex	Band frequency (cm^{-1})	Assignment	10 Dq (cm^{-1})	Racah's parameters (cm^{-1})	L.F.S.E. (k cal. mol^{-1})	$\chi_g \times 10^6$ (c. g. s.)
$Ir(AGDEA)_2Cl_3$	17391	$^3T_{1g} \leftarrow {}^1A_{1g}$	30266	B = 361	104.3	−39.7
	21276	$^3T_{2g} \leftarrow$		C = 4292		
	25974	$^1T_{1g} \leftarrow$				
	31746	$^1T_{2g} \leftarrow$				
$Ir(AGBA)_2Cl_3$	17241	$^3T_{1g} \leftarrow {}^1A_{1g}$	30089	B = 356	103.3	−36.7
	21276	$^3T_{2g} \leftarrow$		C = 4283		
	25806	$^1T_{1g} \leftarrow$				
	31496	$^1T_{2g} \leftarrow$				
$Pt(PGDEA)Cl_4$	21276	$^3T_{1g} \leftarrow {}^1A_{1g}$	36237	B = 451	128.1	−72.9
	23000	$^3T_{2g} \leftarrow$		C = 4987		
	31250	$^1T_{1g} \leftarrow$				
	38462	$^1T_{2g} \leftarrow$				
$Pt(AGDEA)Cl_4$	21053	$^3T_{1g} \leftarrow {}^1A_{1g}$	35627	B = 371	128.0	−16.9
	25000	$^3T_{2g} \leftarrow$		C = 4858		
	30769	$^1T_{1g} \leftarrow$				
	36697	$^1T_{2g} \leftarrow$				
$Pt(AGCA)Cl_4$	20100	$^3T_{1g} \leftarrow {}^1A_{1g}$	32807	B = 369	123.0	−12.3
	22222	$^3T_{2g} \leftarrow$		C = 4236		
	28571	$^1T_{1g} \leftarrow$				
	34482	$^1T_{2g} \leftarrow$				
$Pt(AGBA)Cl_4$	20000	$^3T_{1g} \leftarrow {}^1A_{1g}$	32554	B = 364	122.4	+2.1
	23000	$^3T_{2g} \leftarrow$		C = 4185		
	28369	$^1T_{1g} \leftarrow$				
	34188	$^1T_{2g} \leftarrow$				
$Pt(AGIA)Cl_4$	17544	$^3T_{1g} \leftarrow {}^1A_{1g}$	30189	B = 361	105.5	+0.9
	21276	$^3T_{2g} \leftarrow$		C = 4215		
	25974	$^1T_{1g} \leftarrow$				
	31746	$^1T_{2g} \leftarrow$				

Molecular structures of ligands reveal that carbonyl and azomethine groups are their common donors which display characterisitic IR bands[8,9] at 1740 and 1660 cm^{-1}, 1720 and 1700 cm^{-1}, 1714 and 1684 cm^{-1}, 1710 and 1684 cm^{-1}, and at 1698 and 1634 cm^{-1} in PGDEA, AGDEA, AGCA, AGBA and AGIA respectively. In the IR spectra of complexes these bands occuring at lower frequencies (νc=O at ca. 1683 cm^{-1} and νc=N at ca. 1640 cm^{-1}) indicate biligancy of the ligands through their carbonyl and azomethine groups. Appearance of new bands corresponding to M–O and M–N stretching vibrations[10] at ca. 435 and ca. 463 cm^{-1} respectively in complex spectra supports the above inference. In $Pt(AGBA)Cl_4$ a single broad band observed at 466 cm^{-1} may be attributed to coupled stretching of Pt–O and Pt–N. Disappearance or lowering in frequency and/or intensity of bands, occuring in 814–840 cm^{-1} and 1518–1608 cm^{-1} ranges on account of 1 ; 4 disubstitution and C=C (aromatic) stretching vibrations[8,9] of ligands, in complexes evidently shown molecular rearrangement of ligands possibly from benzenoid to quinonoid structure during complexation. IR frequencies of carbonyl and azomethine groups reveal their sensitiveness[11,12] to the electron repelling ability of para substituents and aromatic nucleus of ligands; these factors may also lead to abnormally high frequency(ies) of characteristic group(s) of ligands.

One or two closely spaced IR bands at ca. 355 cm^{-1} indicate[10] monoligancy of chlorine that is presence of terminal M–Cl bonds in the complexes.

In bis ligand six coordination complexes of Ir(III) with general formula ML_2X_2 (where L and X are bidentate ligand and monodentate halogen respectively) $Ir(AGDEA)Cl_3$ displaying two Ir–Cl infrared bands (390 and 350 cm^{-1}) could be assigned[13] C_{2v} symmetry with chlorines in axial positions but $Ir(AGBA)_2Cl_3$ exhibiting only one Ir–Cl stretching mode (386 cm^{-1}) could be proposed[13] to possess D_{4h} symmetry.

Absorption spectra of d^6 Ir(III) and Pt(IV) complexes display band splitting pattern like low spin complexes of isoelectronic Co(III), Ru(III) and Rh(III) involving octahedrally disposed strong ligand fields[14-17]. In each complex spectrum four ligand field bands have been observed ; first two of which are corresponding to $^3T_{1g} \leftarrow {}^1A_{1g}$ and $^3T_{2g} \leftarrow {}^1A_{1g}$ spin forbidden transitions whereas next two bands could be attributed to $^1T_{1g} \leftarrow {}^1A_{1g}$ and $^1T_{2g} \leftarrow {}^1A_{1g}$ spin allowed transitions. An additional band with very high extinction coefficient observed at ca. 39556 cm^{-1} in all the complexes could be assigned to ligand to metal charge transfer, a^1, $T_{1u} \leftarrow {}^1A_{1g}$. The correctness of band assignments is supported by the fact that these ligand field bands show the expected shift arrangement according to an increasing oxidation numbers, $+4 > +3 > +2$, being obtained in isoelectronic ions. Ligands field parameters (Table 1) have been calculated following the standard procedure[18].

Negative magnetic susceptibilities of complexes revealing their diamagnetic nature are also consistent with absorption spectral inference. Unusual paramagnetism shown by some of the compounds, however, could be attributed to second order Zeeman effect[19] and also to the presence of some spin free state in equilibrium with spin paired configuration. Interestingly, spin free estate could not exist in equilibrium.

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An application of Rota operator

(shift invariant operator/Sheffer set/Pincherle derivative)

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ABSTRACT The Appell Cross-Sequence $C_n^{\nu}(x\,;\,a)$ relative to the invertible shift invariant operator $S=\exp\{\nu(I-E)/a\}$, where E is the shift operator defined as $Ef(x)=f(x+1)$, ν is an integer and a is a non-zero real number, is studied with the help of finite operator calculus developed by Rota *et al.* We obtain binomial identities, generating relation, recurrence relations and several other results for the polynomials $C_n^{\nu}(x\,;\,a)$.

The purpose of present investigation is to study a general class of polynomials $C_n^{\nu}(x\,;a)$ which form a Sheffer set with respect to delta operator $Q=\{(I-E)/a\}$ and the invertible shift invariant operator $S=\exp(\nu Q)$. For special values of ν and a, $C_n^{\nu}(x\,;\,a)$ include as special cases the Charlier polynomials studied by Charlier[1] and the sequence $\phi_n(x)$ due to Carlitz[2]. We make a systematic use of the finite operator calculus developed by Rota *et al.*[3] to investigate this set. We obtain generating relation, recurrence relations and other properties with the help of basic set $q_n(x\,;\,a)$.

For convenience, we shall abbreviate $C_n^{\nu}(x\,;\,a)$ and $q_n(x\,;\,a)$ by $C_n^{\nu}(x)$ and $q_n(x)$, respectively, and a change in any parameter will be indicated by writing that parameter.

Basic sequence: Let $q_n(x)$ denote the basic sequence for the Sheffer set $C_n^{\nu}(x)$ then since the delta operator $Q=\{(I-E)/a\}$, a generating relation for $q_n(x)$ is given[3] by

$$\sum_{n=0}^{\infty} q_n(x)\,\frac{t^n}{n!}=\exp\{xq^{-1}(t)\} \tag{1}$$

where $q^{-1}(t)$ is the formal power series inverse to $q(t)$. Next $Q=q(t)$, $q(t)$ is the indicator of the delta operator. In view of the fact that

$$q(t)=\frac{1-e^t}{a} \tag{2}$$

and

$$q^{-1}(t)=\log(1-at), \tag{3}$$

The above generating relation can be written as

$$\sum_{n=0}^{\infty} q_n(x)\,\frac{t^n}{n!}=(1-at)^x \tag{4}$$

Finally it simplifies to yield

$$q_n(x)=\frac{x!\,(-a)^n}{(x-n)!} \tag{5}$$

By Rota *et al.*[3] we can write

$$q_n(x)=-axq_{n-1}(x-1) \tag{6}$$

and finally

$$q_n(x)=(-a)^n\,(x)_n \tag{7}$$

where $(x)_n=x(x-1)\ldots(x-n+1)$, $n\geq 0$.

Moreover, since the sequence $q_n(x)$ is of binomial type, it satisfies the binomial identity

$$q_n(x+y)=\sum_{r=0}^{n}\binom{n}{r}q_r(x)\,q_{n-r}(y) \tag{8}$$

which, in view of (7), gives

$$(x+y)_n=\sum_{r=0}^{n}\binom{n}{r}(x)_r\,(y)_{n-r} \tag{9}$$

Polynomial $C_n^{\nu}(x)$ as a Sheffer set: Here we study the set $C_n^{\nu}(x)$ as a Sheffer set relative to shift

invariant operator $S=\exp\{\nu(I-E)/a\}$ the delta operator is $Q=\{I-E)/a\}$. Indeed the basic sequence is $q_n(x)$. Now since $C_n^{\nu}(x)$ is a Sheffer set relative to S, we have from Rota *et al.*[3]

$$C_n^{\nu}(x) = S^{-1}\, q_n(x)$$
$$= \exp\{-\nu\,\frac{(I-E)}{a}\}\, q_n(x) \qquad (10)$$

From the definition of Sheffer set

$$QC_n^{\nu}(x) = nC_{n-1}^{\nu}(x) \qquad (11)$$

and consequently

$$Q^p\, C_n^{\nu}(x) = (n)_p\, C_{n-p}^{\nu}(x) \qquad (12)$$

The binomial theorem for Sheffer polynomial yields the following identity

$$C_n^{\nu}(x+y) = \sum_{r=0}^{n}\binom{n}{r} C_r^{\nu}(x)\, q_{n-r}(y) \qquad (13)$$

A generating relation for this Sheffer set is given by

$$\sum_{n=0}^{\infty} C_n^{\nu}(x)\frac{t^n}{n!} = \frac{1}{s(q^{-1}(t))}\exp(x\, q^{-1}(t))$$

where $s(t) = \exp\{-\nu(1-e^t)/a\}$ is the indicator of S and $q^{-1}(t)$ is the same as defined earlier, so that

$$s(q^{-1}(t)) = e^{\nu t}.$$

Thus the generating relation turns out to be

$$\sum_{n=0}^{\infty} C_n(x)\frac{t^n}{n!} = e^{-\nu t}\,(1-at)^x \qquad (14)$$

It is fairly easy to observe that

$$C_n^{\nu}(x) = \sum_{r=0}^{n}(-a)^r\ (-\nu)^{n-r}\, r!\binom{n}{r}\binom{x}{r} \qquad (15)$$

and

$$C_n^{\nu}(x) = \frac{x!\,a^n}{(x-n)!}\,\phi(-n\,;\,x-n+1;\,\nu/a) \qquad (16)$$

$C_n^{\nu}(x)$ as a Cross-Sequence : It is evidently seen that $P^{-\nu} = \exp\{-\nu\,(I-E)/a\}$ forms a one-parameter group of shift invariant operator and for the basic sequence $q_n(x)$, the relation

$C_n^{\nu}(x) = P^{-\nu}\, q_n(x)$ holds.

Thus $C_n^{\nu}(x)$ forms a Cross-sequence[3]

$$C_n^{\nu'}(x) = C_n^{\nu}(x)$$

and we have the identity

$$C_n^{(\nu+\sigma)}(x+y) = \sum_{r=0}^{n}\binom{n}{r} C_r^{(\nu)}(x)\, C_{n-r}^{(\sigma)}(y) \qquad (17)$$

holds for all ν and σ and for any x and y.
The substitution $\nu = -\sigma$ in (17) gives

$$q_n(x+y) = \sum_{r=0}^{n}\binom{n}{r} C_r^{\nu}(x)\, C_{n-r}^{(-\nu)}(y) \qquad (18)$$

Recurrence relations : Here, we derive a recurrence relation for $C_n^{\nu}(x)$ using Pincherle derivative defined on the Algebra of all shift invariant operators. Recall[3] that Pincherle derivative of an operator T is defined as $T' = TX - XT$, where X is the multiplication operator defined on P as X, $X : p(x) \to xp(x)$. We begin with

$$\{\exp(-\nu(I-E)/a\}'\, f(x) = \{\exp(-\nu\,(I-E)/a)x - X\exp(-\nu\,(I-E)/a)\}\, f(x)$$

and thus

$$\{\exp(\frac{-\nu}{a}\,(I-E)\}\, xf(x) = \{\exp(\frac{-\nu}{a}\,(I-E))\}'\, f(x) + x\,\{\exp(\frac{-\nu}{a}\,(I-E))\}\, f(x)$$

If we put $f(x) = (-a)^n\,(x-1)_{n-1}$ and simplify a little more, we obtain

$$C_{n+1}^{\nu}(x) + C_n^{\nu}(x) + ax\, C_n^{\nu}(x-1) = 0 \qquad (19)$$

we can also derive

$$(i)\ C_n^{\nu}(x+1) - C_n^{\nu}(x) + an\, C_{n-1}^{\nu}(x) = 0 \qquad (20)$$

$$(ii)\ C_{n+1}^{\nu}(x) + (\nu - an + ax)\, C_n^{\nu}(x) - \nu\, an\, C_{n-1}(x) = 0 \qquad (21)$$

$$(iii)\ C_{n+1}^{\nu}(x) + a(x-n)\, C_n^{\nu}(x) - \nu\, C_n^{\nu}(x+1) = 0 \qquad (22)$$

$$(iv)\ \nu\, C_n^{\nu}(x+1) + (an - ax + \nu)\, C_n^{\nu}(x) + ax\, C_n^{\nu}(x-1) = 0 \qquad (23)$$

Eigen function expansion : Let $W : C_n^{\nu}(x) \to (x)_n$ be the umbral operator sending $C_n^{\nu}(x)$ to $(x)_n$. Then

$$(C_r^{\nu}(x),\, C_n^{\nu}(x)) = [(WC_r^{\nu})\, Q\, (SC_n^{\nu}(x))]_{x=0} \qquad (24)$$

which is the natural inner product associated with the Sheffer set $C_n^{\nu}(x)$.

We have the following

Preposition : The bilinear form $C_r^{\nu}(x)$, $C_n^{\nu}(x))$ defined by (24) on the vector space of all polynomials is a positive definite inner product.

Proof. To establish this it is sufficient to show that

$$[(WC_r^{\nu})\ Q\ (SC_n^{\nu}(x)]_{x=0}$$
$$= [(Q)_r\ q_n(x)]_{x=0}$$
$$= n!\ \delta_{rn},$$ which completes the proof.

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A counter example in fixed point theory on closed sets through abstract cones

(regular cone/monotone/sub additive)

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ABSTRACT A counter e ample is given to show that certain results of Eisenfeld and Lakshmikantam are false.

Let E be a real Banach space. A set $K \subset E$ is said to be a cone if (i) K is closed, (ii) if $x,y \in K$, then $ax + by \in K$ for all $a,b \geqslant 0$ and (iii) if x and $-x$ are in K, then $x=\theta$, the zero element of E. For x,y in E, we say $x \geqslant y$ if $x-y \in K$. A sub set y of E is said to be K-bounded if there exists Z in E such that $y \leqslant Z$ for all y in Y. The cone K is said to be a regular cone if every increasing sequence x_n $(x_1 \leqslant x_2 \leqslant \ldots)$ in E which is K-bounded converges with respect to the norm in E.

A map $f : K \to K$ is said to be (i) monotone if $f(x) \leqslant f(y)$ for $x \leqslant y$ and (ii) subadditive if $f(x+y) \leqslant f(x) + f(y)$.

A K-normed linear space is a linear space X equipped with a cone valued norm, (*i.e.*) for x,y in X and scalar a, (i) $\| x \| \in K$, (ii) $\| ax \| = |a| \, \| x \|$, (iii) $\| x+y \| \leqslant \| x \| + \| y \|$ and (iv) $\| x \| = \theta$ iff $x=0$, the zero in X.

A complete K-normed linear space is called a K-Banach space.

Eisenfeld and Lakshmikantam[1] proved the following three results :

Result (1): Let K be a regular cone and $f : K \to K$ be monotone and either f is upper semi continuous from the right 'or' lower semi continuous from the left.

Suppose :

For every v in K the eqn. $f(u)+v=u$ has atmost one solution in K. (1)

For every v in K, $\exists\, u \geqslant v \ni f(u) + v \leqslant u$. (2)

Then $\sum_{n=0}^{\infty} f^n(v)$ converges for $v \in K$.

Result (2) : Let K be a regular cone and suppose $f : K \to K$ is monotone and subadditive. Assume $\exists\, u_0 > \theta \ni$ (i) $u_0 > f(u_0)$;

(ii) $\lim_n f\left(\frac{u_0}{n}\right) = \theta$

(iii) θ is the unique fixed point of f in $<\theta, u_0>$. Then the following is true :

(a) f is continuous

(b) conditions (1) and (2) of result (1) are satisfied

(c) $\sum_{n=0}^{\infty} f^n(v)$ converges for every $v \in K$ and the map $r(v) = \sum_{n=0}^{\infty} f^n(v)$ is monotone, subadditive and continuous.

Result (3) : Let X be a K-Banach space where the cone K is regular. Let D be a closed subset of X and $G : D \to X$ a continuous function. Let $f : K \to K$

be a monotone, subadditive and let $\exists\, u_0 > \theta \ni$ (i) $u_0 > f(u_0)$; (ii) $\lim_n f(\frac{u_0}{n}) = \theta$; (iii) θ is the unique fixed point of f in the segment $< \theta, u_0 >$. Suppose for arbitrary x in D ωy in D can be found in accordance with the following conditions :

(a) y depends continuously on x.

(b) $\| y-x \| \leqslant \| Gx \|$ and $y = x$ iff $\| Gx \| = \theta$

(c) $\| Gy \| \leqslant f (\| Gx \|)$.

Then for abrbitrary x_0 in D, the sequence $\{x_n\}$, where x_{n+1} is the y corresponding to x_n, converges to a vector $Z \ni \| GZ \| = \theta$ and $\| x_n - Z \| \leqslant f(\|Gx_n\|)$, $n = 1,2, \ldots$

Unfortunately, these three results are false in view of the following example ;

Example: Let $X = IR$, and $K = [0,\infty)$. Then X is a K-Banach space.

Define $f : K \to K$ by $f(t) = \frac{t}{1+t}$.

Then $\sum_{n=0}^{\infty} f^n(v)$ does not converge for any non zero v in K, which shows that results (1) and (2) are false.

Write $D = \{a_1, a_2, \ldots\}$ where $a_n = \sum_{m=1}^{n} \frac{1}{m}$

Define $g : D \to D$ by $g(a_n) = a_{n+1}$

with $y = g(x)$ and $G = I - g$, all the hypothesis of the result (3) is satisfied but not the conclusion.

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Coincidence theorems on 2-metric spaces

(mapping/coincidence theorem/2-metric spaces)

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ABSTRACT Coincidence theorems for three mappings on an arbitrary set having values in a 2-metric space are proved. Our results extend and unify a number of known coincidence theorems and fixed point theorems for mappings satisfying general contractive conditions.

Throughout this note X will denote a non empty arbitrary set, (Y,d) a 2-metric space[1] and P,S,T, mappings on X with values in (Y,d). Consider the following conditions :

$$d(Px, Py, a) \leqslant k\, d(Sx, Sy, a) \quad (1)$$

for all x,y in X, all a in Y and some $k \in (0,1)$.

$$d(Px,Py,a) \leqslant k \max \{d(Sx,Ty,a), d(Px,Sx,a), d(Py,Ty,a), \tfrac{1}{2}[d(Px,Ty,a) + d(Py,Sx,a)]\} \quad (2)$$

for all x,y in X, all a in Y and some $k \in (0,1)$.

$$d(Px,Py,a) \leqslant kd(Py,Ty,a)\frac{c+d(Px,Sx,a)}{c+d(Sx,Ty,a)} + k'd(Sx,Ty,a) \quad (3)$$

for all x,y in X, all a in y, c a positive number, and k and k' non-negative numbers with $0<k+k'<1$.

Using the Banach contraction principle, Goebel[2] proved an interesting coincidence theorem for a pair of mappings on an arbitrary set with values in a metric space satisfying condition analogous to (1). Recently Goebel's result has been extended and unified by many other workers[3-6].

Singh and Virendra[4] proved coincidence theorems under the conditions (2) and (3) with $S=T$, and derived some known fixed point theorems on 2-metric spaces.

The aim of this work is to obtain coincidence theorems under the conditions (2) and (3). As a consequence, we also derive some fixed point theorems which extend and unify the results of otners[4,5,7-10].

We shall need the following Lemma of Singh[11].

Lemma : Let $\{y_n\}$ be a sequence in a 2-metric space Y. If there exists $k \in (0,1)$ such that

$$d(y_n, y_{n+1}, a) \leqslant kd(y_{n-1}, y_n, a)$$

for all n and $a \in Y$, then $\{y_n\}$ is a Cauchy sequence.

Coincidence Theorems

Theorem 1 : Let P,S and T be mappings from X to Y satisfying the condition (2) and the following ;

(I) $P(X) \subseteq S(X) \cap T(X)$; (II) $S(X) \cap T(X)$ is a complete subspace of Y.

Then (i) P and S have a coincidence point ; (ii) P and T have a coincidence point ; (iii) P,S and T have a coincidence point provided P is one-one.

Proof : Pick $x_0 \in X$. Construct, in view of (I), a sequence $\{x_n\}$ of points of X as follows :

$$Sx_{2n+1} = Px_{2n},\ Tx_{2n+2} = Px_{2n+1},\ n=0,1,2,\ldots$$

By (2),

$$d(Px_{2n+1}, Px_{2n+2}, a) \leqslant k \max \{d(Px_{2n}, Px_{2n+1}, a), d(Px_{2n}, Px_{2n+1}, a), d(Px_{2n+1}, Px_{2n+2}, a), \tfrac{1}{2}[d(Px_{2n+1}, Px_{2n+1}, a) + d(Px_{2n}, Px_{2n+2}, a)]\},$$

which gives, as in Singh[10]

$$d(Px_{2n+1}, Px_{2n+2}, a) \leqslant kd(Px_{2n}, Px_{2n+1}, a).$$

Similarly,

$$d(Px_{2n+2}, Px_{2n+3}, a) \leqslant kd(Px_{2n}, Px_{2n+1}, a)$$

In general

$$d(Px_{n+1}, Px_{n+2}, a) \leqslant kd(Px_n, Px_{n+1}, a) \text{ for all } a \text{ in } Y.$$

So, by the above Lemma, $\{Px_n\}$ is a Cauchy sequence, and has a limit in $S(X) \cap T(X)$. Call it u. Then there exists a z in X such that $Sz=u$.

Again by (2),

$$d(Pz, Px_{2n+2}, a) \leqslant k \max \{d(Sz, Px_{2n+1}, a), d(Pz, Sz, a), d(Px_{2n+1}, Px_{2n+2}, a), \tfrac{1}{2}[d(Pz, Px_{2n+1}, a) + d(Px_{2n+2}, Sz, a)]\}.$$

Letting $n \to \infty$, we have

$$d(Pz, Sz, a) \leqslant k \max \{d(Sz, Sz, a), d(Pz, Sz, a), 0, \tfrac{1}{2}[d(Pz, Sz, a) + d(Sz, Sz, a)]\},$$

giving

$$d(Pz, Sz, a) \leqslant kd(Pz, Sz, a),$$

a contradiction, since a is arbitrary. So $Pz=Sz$.

Similarly, there exists a y in X such that $Py=Ty$. Clearly, $Pz=Sz=u=Ty=Py$. So $z=y$, since P is one-one.

This Proves (iii).

Remark : The main result of Singh and Virendra[4] is obtained when $S=T$ in Theorem 1 above.

Theorem 2 : Let P,S and T be mappings from X to Y satisfying the conditions (3), (I) and (II). Then the conclusions (i) – (iii) of Theorem 1 hold.

Proof : Pick $x_0 \in X$, and construct a sequence $\{Px_n\}$ of points of Y as in the proof of Theorem 1. By (3),

$$d(Px_{2n-1}, Px_{2n}, a) \leqslant kd(Px_{2n-1}, Px_{2n}, a) \frac{c+d(Px_{2n-2}, Px_{2n-1}, a)}{c+d(Px_{2n-2}, Px_{2n-1}, a)} + k' d(Px_{2n-2}, Px_{2n-1}, a),$$

that is

$$d(Px_{2n-1}, Px_{2n}, a) \leqslant hd(Px_{2n-2}, Px_{2n-1}, a) \quad (4)$$

where $h=k'/(1-k) \in (0,1)$.

Again by (3),

$$d(Px_{2n+1}, Px_{2n}, a) \leqslant kd(Px_{2n}, Px_{2n-1}, a) \frac{c+d(Px_{2n+1}, Px_{2n}, a)}{c+d(Px_{2n}, Px_{2n-1}, a)} + k' d(Px_{2n}, Px_{2n-1}, a).$$

This can be written as

$$d_{2n} \leqslant kd_{2n-1} \frac{c+d_{2n}}{c+d_{2n-1}} + k'd_{2n-1},$$

where $d_m \equiv d(Px_m, Px_{m+1}, a)$.

So $$d_{2n}\left(1-\frac{kd_{2n-1}}{c+d_{2n-1}}\right) \leqslant \left(\frac{kc}{c+d_{2n-1}} + k'\right) d_{2n-1}$$

that is $$d_{2n} \leqslant \frac{kc+k'c+k'd_{2n-1}}{c+d_{2n-1}-kd_{2n-1}} d_{2n-1}$$

that is $d_{2n} \leqslant \frac{\alpha}{\beta} d_{2n-1}$, where $\alpha = kc+k'(c+d_{2n-1})$ and $\beta = c+(1-k)d_{2n-1}$.

Let, if possible, $\alpha > \beta$. Then

$$kc + k'(c+d_{2n-1}) > c+(1-k)d_{2n-1},$$

that is $(1-k)d_{2n-1} < k'd_{2n-1} - c(1-k-k')$,

that is $d(Px_{2n-1}, Px_{2n}, a) \leqslant hd(Px_{2n-1}, Px_{2n}, a)$, a contradiction to $h \in (0,1)$. So $\alpha \leqslant \beta$, and hence

$$d(Px_{2n+1}, Px_{2n}, a) \leqslant d(Px_{2n}, Px_{2n-1}, a) \quad (5)$$

In view of (4) and (5), we have

$$d(Px_{2n+1}, Px_{2n+2}, a) \leqslant h^{n+1} d(Px_0, Px_1, a)$$

and

$$d(Px_{2n+2}, Px_{2n+3}, a) \leqslant h^{n+1} d(Px_0, Px_1, a).$$

Now it is not difficult to see that $\{Px_n\}$ is a Cauchy sequence. So it has limit in $S(X) \cap T(X)$. Call it u. Then there exists a z in X such that $Sz=u$. By (3),

$$d(Pz, Px_{2n+2}) \leqslant kd(Px_{2n+2}, Px_{2n+1}, a) \frac{c+d(Pz, Sz, a)}{c+d(Sz, Px_{2n+1}, a)} + k' d(Sz, Px_{2n+1}, a).$$

Letting $n \to \infty$ we have

$d(Pz, Sz, a) \leqslant 0$, proving $Pz=Sz$.

Conclusions (ii) and (iii) follow easily.

Remark : Theorem 3 of Singh and Virendra[4] is obtained when $S=T$ in Theorem 2 above.

Fixed Point Theorems

Throughout this section, assume that $X=Y$ and that the mapping conditions considered in the previous sections should be assumed to hold for every $x,y \in Y$.

Theorem 3 : Let $P,S,T : Y \to Y$ satisfy the conditions (2), (I), (II) and the following :

(III) $PS=SP$ and $PT=TP$.

Then P,S and T have a unique common fixed point.

Proof : In view of Theorem 1, there exist u,z,y in Y so that

$$Px_n \to u, Pz=Sz=u=Py=Ty.$$

By (III),

$$Tu=TPz=PTz=Pu$$

and $Su=SPz=PSz=Pu$.

By (2),

$$\begin{aligned} d(Pu,u,a) &= d(Pz,Pu,a) \\ &\leq k \max \{d(Sz,Tu,a), d(Pz,Sz,a), \\ &\quad d(Pu,Tu,a), \tfrac{1}{2}[d(Pz,Tu,a)+d(Pu, \\ &\quad Sz,a)]\} = k \max \{d(u,Pu,a),0,0,d(u, \\ &\quad Pu,a)\} = k\, d(Pu,u,a), \end{aligned}$$

implying $Pu=u$. Thus $Pu=Su=Tu=u$. Uniqueness of u as a common fixed point of P, S and T follows easily.

Remark 1 : This theorem improves the result of Singh[10]. In fact, Theorem 3 with continuity condition on S and T is proved in Singh[10]

Remark 2 : Theorem 1 of Singh *et al.*[8] is obtained when $S=T$ and T continuous in the above theorem.

Theorem 4 : Let $P,S,T : Y \to Y$ satisfy the conditions (3), (I), (II) and (III). Then P,S and T have a unique common fixed point.

Proof : It may be completed following the above proof.

Remark 1 : Theorem with continuity condition on P,S and T is proved in Ram[9].

Remark 2 : Corollary 2 of Singh and Virendra[4] is obtained when $S=T$ in the above theorem.

Applications : In this section we apply Theorems 3 and 4 to prove two results what may be called 'fixed point theorems on product spaces'.

Theorem 5 : Let Y be a complete 2-metric space, and P,S and T be mappings from the product space $Y \times Y$ to Y such that

$$P(Y \times \{x\}) \subseteq S(Y \times \{x\}) \cap T(Y \times \{x\}) \quad (6)$$

$S(Y_x \{x\}) \cap T(Y_x \{x\})$ is a closed subspace of Y (7)

$$P(S(x,y),y)=S(P(x,y),y), P(T(x,y),y) = T(P(x,y),y) \quad (8)$$

for all x,y in Y. If there exists $k \in (0,1)$ such that

$$\begin{aligned} d(P(x,y), P(x',y'), a) \leq k \max \{&d(S(x,y), \\ &T(x',y'), a), d(P(x,y), S(x,y), a), \\ &d(P(x',y'), T(x',y'), a), \\ &\tfrac{1}{2}[d(P(x,y), T(x',y'), a), + d(P('x,y'), \\ &S(x,y), a)]\} \end{aligned} \quad (9)$$

for all x,y,x',y',a in Y, then there exists exactly one point b such that $P(b,y)=S(b,y) = T(b,y) = b$ for all y in Y.

Proof : By (9),

$$\begin{aligned} d(P(x,y), P(x',y), a) \leq k \max \{&d(S(x,y), \\ &T(x',y), a), d(P(x,y), S(x,y), a), d(P \\ &(x',y), T(x',y), a), \tfrac{1}{2}[d(P(x,y), T(x',y), a) \\ &+ d(P(x',y), S(x,y), a)]\} \end{aligned}$$

for every $x, x', y, a \in Y$. Therefore, by Theorem 3, for each y in Y, there exists one and only one $x(y)$ such that

$$P(x(y),y) = S(x(y),y) = T(x(y),y) = x(y).$$

For every $y,y', a \in Y$ we have by (9),

$$\begin{aligned} d(x(y), x(Py'), a) = d(x(x(y),y), P(x(y'),y'), \\ a) \leq kd(x(y), x(y'), a) \end{aligned}$$

and consequently $x(y) = x(y')$ since a is arbitrary. Hence $x(\cdot)$ is some constant $b \in Y$ such that

$$P(b,y) = S(b,y) = T(b,y) = b \text{ for all } y \text{ in } Y.$$

Theorem 6 : Let y be a complete 2-metric space, and P,S and T be mappings from the product space $Y \times Y$ to Y satisfying the conditions (6), (7), (8) and the following :

$$d(P(x,y), P(x',y'), a) \leqslant kd(P(x',y'), T(x',y'), a) \frac{c + d(P(x,y), S(x,y), a)}{c + d(S(x,y), T(x',y'), a)} + k' d(S(x,y), T(x',y'), a)$$

for all x, y, x', y', a in Y, where k, k' are as in Theorem 2.

Then $P(b,y) = S(b,y) = T(b,y) = b$ for all y in Y.

Proof: It may be completed following Theorems 4 and 5.

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Metal film strain gauges

(transducers/metal film/strain gauges)

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ABSTRACT Strain gauge factor is an important parameter from the point of view of development and fabrication of strain gauge pressure transducers. In case of metal film strain gauges, thermal expansion, differential thermal stress and partial specular scattering at the surface's play an important role in determining its behaviour. These parameters may, therefore, be fruitfully exploited in the development of metal film strain gauges.

Metal film strain gauges are practically more compatible as compared to metal foil and metal wire strain gauges. In addition, they can be used over larger temperature ranges as compared to semiconducting film strain gauges (having larger strain sensitivity) because of their lesser sensitivity against temperature variations. Thin film strain gauges are usually formed by depositing films at suitable insulating and flexible substrates which serve as a media for connecting strain gauges to the specimen under test. Thus, it is promising to discuss metal film strain gauges from the point of view of optimal stable behaviour.

A good strain gauge should offer a high change in resistance on being strained. Typically, the contribution to change in resistance arises on two counts ; one due to dimensional changes resulting from strain and other due to strain sensitivity of the resistivity which is physical contribution[1]. In case of metal film gauges, this physical contribution will also depend on the surface scattering contribution to the film's resistivity. In fact, for the metalic films, the surface scattering contribution usually dominates the bulk or background scattering contribution. Apart from the surface scattering contribution, grain boundary scattering also contributes to the total film resistivity for a polycrystalline film if grain sizes are comparable to or less than the bulk mean free path length[2]. In this paper, we critically examine the behaviour of a thin metal film strain gauge with a view to look for its optimal performance.

Most of metal films are known to exhibit a gauge factor $(\frac{1}{R}\frac{dR}{dT})$ variation which decreases and reaches a minimum with decrease in thickness and then rises as the film becomes discontinuous. Limiting the discussion to continuous metal films with sufficiently large grain sizes, one has to focus attention to strain sensitivity of metallic films with dominant surface scattering. This case has been investigated by several workers[3-7]. Since the surface scattering in thin metallic films is sensitive to thermal expansion and differential thermal stress, these effects cannot be overlooked in general. In a practical situation, metallic films are usually in contact with an insulating substrate on one surface while the other surface is exposed to vacuum/atmosphere or is covered with a

protective coating[4]. Thus the empirical specularity parameter is more likely to be different on the two film surfaces. Taking all these into consideration the longitudinal γ_{FL} and transverse γ_{FT} gauge factors are given as,

$$\gamma_{FL/FT}=\gamma_{OL/OT}+\frac{\left(\frac{\beta_f(k,p_1,p_2)}{\beta_0}-1\right)\left(\eta-\mu_f\,\frac{1-\mu_s}{1-\mu_f}\right)}{\left[1+\frac{\alpha_{lf}}{\beta_0}\left\{1+\frac{2\mu_f\,(1-\frac{\alpha_{ls}}{\alpha_{lf}})}{1-\mu_f}\right\}\right]}$$

Here β_f is the TCR of the metallic films, (α_{lf}, μ_f) and (α_{ls}, μ_s) are the expansion coefficients and Poisson's ratio of the film and the substrate material respectively, p_1, p_2 are specularity parameters at the two surfaces and k is the reduced thickness.

A sample calculation of γ_{FL} according to above equation showing the effect of expansion and partial specular reflections at the two surfaces are given in the Table 1.

From the Table it may be noted that film expansion, differential thermal stress and varying surface conditions at the two surfaces make significant contribution to the strain sensitivity of the metal film. It may be noted that the contribution of these factors is more for metal films of lower reduced thickness which may be achieved in films of metals having larger mean free path lengths for the conduction electrons or in case the metal films are used in low temperature environments. Since the expansion effects and differing surface conditions tend to increase the gauge factors, these parameters may be fruitfully exploited in the development and fabrication of thin metal film strain gauges to optimise their performance.

TABLE 1

Gauge factor values for supported alkali metal films as a function of reduced thickness

Reduced thickness	without expansion	with expansion		
	$\alpha_{lf}=\alpha_{ls}=0$	$\frac{\alpha_{lf}}{\beta_0}=0.05$	$\frac{\alpha_{ls}}{\alpha_{lf}}=0.05$	
k	$p_1=p_2=0$	$p_1=p_2=0$	$p_1=0\ p_2=\frac{1}{2}$	$p_1=0\ p_2=\frac{1}{2}$
0.1	1.6368	1.7310	1.8143	1.8791
0.5	1.7971	1.8619	1.8170	1.9648
1.0	1.8904	1.9380	1.9801	1.0183
2.0	1.9918	2.0208	2.0516	2.0811

$\eta=1.18$, $\mu_f=0.38$, $\mu_s=0.02$

$f(k, p_1, p_2)$ values are used as given in ref. 4.

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Efficacy of foliar systemic insecticides against green peach aphid, *Myzus persicae* Sulzer on potatoes

(*Myzus persicae*/oxidemeton-methyl/dimethoate/monocrotophos)

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ABSTRACT Field evaluation of six foliar systemic insecticides against *Myzus persicae* reveal that all the insecticides *viz*. oxydemeton-methyl, dimethoate, phosphamidon, thiometon, formothion and monocrotophos as sprays @ 325 g/ha are quite effective in controlling the aphid, *Myzus persicae* Sulzer on potato crop. However, oxydemeton-methyl, monocrotophos and dimethoate registered consistently better control.

For producing healthy seed stock of potatoes, it is essential to control the potential aphid vector like *Myzus persicae* Sulzer which is mainly responsible for the field spread of important potato viruses like leaf roll and virus 'Y'. Earlier literature on the evaluation of systemic insecticides revealed that phorate (Thimet 10 G), disulfoton (Disyston 5 G), oxydemeton-methyl (Metasystox 25 EC) and dimethoate (Rogor 30 EC) are effective against potato aphids[1-4]. The present studies deal with the field evaluation of oxydemeton-methyl and dimethoate (emulsifiable concentrates) alongwith some newer systemic insecticides as sprays against green peach aphid, *Myzus persicae* Sulzer on potato crop.

Field experiments were conducted at the farm of Central Potato Research Station, Jalandhar (Punjab) during spring seasons of 1979 and 1980 for evaluating the relative efficacy of six foliar systemic insecticides *viz* : oxydemeton-methyl 25 EC, dimethoate 30 EC, phosphamidon 100, thiometon 25 EC, formothion 25 EC and monocrotophos 40 EC applied @ 325 g active ingredient/ha against the green peach aphid, *Myzus persicae* Sulzer. For experimentation disease-free seed of potato cv. Kufri Chandramukhi was used. There were seven treatments including control which were replicated thrice in randomized block designs. Recommended agronomical practices of the region were followed for raising the crops.

The aphicidal effect of insecticides was evaluated by recording the population of *M. persicae* on 34 plants (100 compound leaves) per plot on second, fifth and ninth day after spraying and the data recorded were analysed after $\sqrt{X+1}$ transformation[5].

An examination of 1979 data (Table 1) reveals that all the insecticides were significantly superior over check in controlling the aphids in all the three observations. However, in the first observation (2 days after spray application) monocrotophos, oxydemeton-methyl, dimethoate, phosphamidon and thiometon were relatively more effective than formothion. In second observation (5 days after application) all the insecticides were equally effective while at the time of third observation (9 days after

spraying) oxydemeton-methyl and dimethoate were superior over phosphamidon, thiometon and formothion being at par with monocrotophos.

TABLE 1

Efficacy of foliar systemic insecticides against green peach aphid, *Myzus persicae* Sulzer on potato crop during spring seasons of 1979 and 1980.

Treatment	Aphid populations (mean of 3 replications)/100 compound leaves					
	Days after spray treatment during 1979			Days after spray treatment during 1980		
	2	5	9	2	5	9
Oxydemeton-methyl (Metasystox 25 EC)	3.95 (14.60)	4.91 (23.11)	4.03 (15.24)	1.00 (0.00)	1.00 (0.00)	1.00 (0 00)
Dimethoate (Rogor 30 EC)	2.60 (5.76)	4.10 (15.81)	3.45 (10.90)	1.33 (0.77)	1.00 (0.00)	1.00 (0.00)
Phosphamidon (Dimecron-100)	4.49 (19.16)	6.39 (39.83)	7.89 (61.25)	1.00 (0.00)	1.33 (0.77)	1.00 (0.00)
Thiometon (Ekatin 25 EC)	3.96 (14.68)	7.21 (50.98)	7.36 (53.17)	1.00 (0.00)	1.00 (0.00)	1.00 (0.00)
Formothion (Anthio 25 EC)	6.07 (35.85)	5.61 (30.47)	7.18 (50.55)	1.00 (0.00)	2.44 (4.95)	1.00 (0.00)
Monocrotophos (Azodrin 40 EC)	2.05 (3.20)	6.07 (35.85)	5.13 (25.32)	1.94 (2.76)	1.33 (0.77)	1.00 (0.00)
Control (water spray)	23.79 (564.96)	17.14 (292.78)	19.88 (394.21)	8.63 (73.50)	8.39 (69.39)	2.05 (3.20)
S. Em (±)	0.79	1.18	0.92	0.61	0.46	0.26
C. D. (0.05)	2.43	3.64	2.83	1.88	1.42	0.57

Figures in parenthesis under aphid population column are di-transformed original values.

The data of 1980 (Table 1) show that all the insecticides were significantly superior over control in all the observations. However, there was no significant difference among themselves, except in the case of formothion at the time of second observation. Results of the two years experiments suggest that all the six foliar systemic insecticides are significantly superior over check (water spray) in controlling *M. persicae*. It is further seen that oxydemeton-methyl, monocrotophos and dimethoate ECs registered consistently better control than the rest of the insecticides. Hence, seed based sprayings with any of the foliar systemic insecticides (preferably with oxydemeton-methyl/dimethoate/monocrotophos) are suggested for controlling *M. persicae* on seed potato crops.

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Lindane induced alterations in the protein breakdown and utilization in the selected tissues of fresh water fish, *Tilapia mossambica* (Peters)

(lindane toxicity/protein breakdown/*Tilapia mossambica*)

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ABSTRACT Under *in vivo* lindane toxicity the protein content of the tissues were decreased with a corresponding increase in the neutral protease activity leading to the increment in the tissue total amino acid level. Corraborating with this, there is increment in the alanine and aspartate aminotransferase activity levels. The significance of the alterations have been discussed.

Lindane, an organochlorine insecticide is widely used with merit on crops and in public health programmes. The indiscriminate use of insecticides leads to environmental pollution and affects non target organisms including man. Not much work is reported on the effects of organochlorine insecticides on the metabolic profiles of non-target animals[1-3]. A study was therefore conducted on certain aspects of catabolism and utilization of protein in the brain, liver, muscle and gill tissues of *T. mossambica* under lethal and sublethal concentrations of lindane.

The fish were collected from fresh water tanks in Tirupathi and acclimatized to laboratory conditions. The toxicity evaluation studies were conducted as described by Doudoroff *et al.*[4]. The LC 50, determined by both probit[5] and calculated methods[6], was found to be 0.15 ppm. Estimations were made at lethal and sublethal treatments.

The sucrose soluble and insoluble proteins were estimated by the method of Lowry *et al.*[7], the total ninhydrin positive substances (free amino acid content) by Moore and Stein[8] method and the activity levels of neutral protease and aspartate and alanine aminotransferases by the methods described by Moore and Stein[8] and Reitman and Frankel[9], respectively.

The results indicate that all the selected parameters except proteins have recorded an elevation (Tables 1–3), while the proteins have decreased. The increase or decrease, which is tissue specific, is more under lethal concentration than under sublethal concentration, the changes reaching the maximum at 48 h of exposure.

The liver tissue has recorded higher percentage decrease in soluble protein content followed by brain, muscle and gill. Since, the liver is the seat of metabolism and soluble proteins represent the activity level of enzymes in general[10], the activity of the liver tissue is apparently more affected than the other tissues thereby showing lindane as hepatotoxic. This confirms reports suggesting organochlorides as hepatotoxicants[11,12].

The tissue sucrose insoluble protein content recorded maximum decrement in muscle followed by liver, gill and brain. Since the insoluble protein

TABLE 1

Sucrose soluble protein (SP) and insoluble protein (ISP) contents in selected tissues of Fish exposed to lethal (0.15 ppm) and sublethal (0.05 ppm) concentrations of lindane. Each value is $\bar{x} \pm$ SD of 6 observations. The values in parenthesis are per cent change over control. Values are expressed as mg protein/g wet wt. All values are significant at $P < 0.001$ except a : Not significant b : $P < 0.005$; c : $P < 0.025$; d : $P < 0.01$

Tissue	Control		Experimental											
			Lethal exposure h						Sublethal exposure h					
			12		24		48		12		24		48	
	SP	ISP	SP	ISP	SP	ISP	SP	ISP	SP	ISP	SP	ISP	SP	ISP
Brain	98.93	47.35	101.90[a]	55.60[d]	93.66[a]	42.84[a]	85.53	38.76	100.91[a]	49.12[a]	94.34[a]	46.35[a]	89.89[a]	45.89[a]
	±3.54	±1.92	±3.66	±2.55	±1.89	±1.89	±2.07	±1.78	±3.72	±1.57	±3.68	±1.51	±4.84	±1.49
			(+3.0)	(+17.4)	(-5.3)	(-9.5)	(-13.6)	(-18.2)	(+1.9)	(+3.7)	(-4.6)	(-2.1)	(-9.2)	(-3.1)
Liver	91.10	52.35	72.35[b]	54.79	62.27	50.24[a]	56.11	40.55	84.16[a]	50.73[a]	74.28	48.43[a]	62.12	46.41[c]
	±3.93	±2.00	±1.91	±3.05	±2.20	±2.63	±2.17	±2.93	±3.53	±1.35	±3.46	±1.37	±2.98	±2.09
			(-20.5)	(+23.7)	(-31.6)	(-4.0)	(-38.4)	(-22.6)	(-7.6)	(-3.9)	(-18.4)	(-7.5)	(-31.8)	(-11.3)
Muscle	28.15	64.32	28.95[a]	56.62[d]	25.66[c]	49.65	18.73	44.35	31.13[c]	58.74[d]	27.88[a]	51.66	22.91[b]	48.28
	±0.94	±2.87	±2.12	±2.36	±1.41	±1.58	±2.17	±1.20	±1.47	±1.81	±1.39	±1.64	±1.04	±1.11
			(+2.8)	(-11.9)	(-8.8)	(-22.8)	(33.4)	(-31.0)	(+10.6)	(-8.6)	(-1.0)	(-19.7)	(-18.5)	(-24.9)
Gill	23.13	56.33	23.76[a]	57.87[a]	21.45[c]	58.05[a]	18.62	45.46	23.45[a]	56.37[a]	22.72[a]	47.13[a]	20.58[c]	42.57[a]
	±0.89	±2.34	±1.55	±1.36	±1.75	±1.29	±0.81	±1.41	±1.27	±0.96	±2.75	±0.79	±1.68	±1.43
			(+2.7)	(+2.6)	(-7.2)	(+3.0)	(-19.5)	(-19.3)	(+1.2)	(+0.6)	(-1.8)	(+1.2)	(-11.1)	(-6.7)

TABLE 2

Total free amino acid content (A A) (Ninbydrogen positive substances) (μ moles of tyrosine formed/g wet wt.) and levels of neutral protease (NP) activity (μ moles of tyrosine formed/mg. protein/h) in selected tissues of fish exposed to lethal (0.15 ppm) and sublethal (0.05 ppm) concentrations of lindane. Each value is $\bar{x} \pm$ SD of 6 observations. The values in parenthesis are per cent change over control. All values are significant at $P < 0.001$ except a : Not significant ; b : $P < 0.005$; c : $P < 0.025$; d : $P < 0.01$

Tissue	Control		Experimental											
			Lethal exposure h						Sublethal exposure h					
			12		24		48		12		24		48	
	AA	NP	AA	NP	AA	NP	AA	NP	AA	NP	AA	NP	AA	NP
Brain	68.55	0.399	102.51	0.579	130.11	0.729	157.53	0.898	92.45	0.516[a]	115.31	0.795	138.22	0.835
	±4.42	±0.032	±8.45	±0.042	±12.50	±0.051	±18.03	±0.134	±11.27	±0.105	±10.91	±0.071	±11.85	±0.111
			(49.60)	(45.40)	(89.70)	(83.2)	(129.90)	(125.10)	(34.90)	(29.30)	(68.30)	(99.20)	(101.80)	(109.30)
Liver	78.50	0.673	128.50	1.092	161.16	1.325	197.83	1.669	116.00	0.989[a]	144.56	1.125	172.30	1.589
	±6.22	±0.045	±4.23	±0.047	±5.94	±0.064	±12.73	±0.095	±8.62	±0.113	±7.89	±0.095	±9.18	±0.111
			(63.70)	(62.30)	(105.30)	(96.70)	(152.00)	(147.90)	(47.80)	(56.90)	(84.10)	(89.70)	(119.50)	(136.10)
Muscle	70.50	0.593	96.33	0.963	120.17	1.095	167.16	1.408	89.66	0.851	118.66	1.103	147.60	1.326
	±4.96	±0.086	±12.34	±0.045	±11.68	±0.049	±15.42	±0.116	±10.29	±0.082	±8.24	±0.094	±15.78	±0.112
			(36.60)	(62.30)	(70.40)	(84.60)	(137.10)	(137.40)	(27.20)	(43.30)	(68.30)	(86.0)	(109.40)	(123.60)
Gill	50.36	0.329	66.48	0.432	82.31	0.521	107.32	0.605	64.57	0.418[a]	80.10	0.512	99.40	0.586
	±4.03	±0.069	±6.46	±0.048	±12.07	±0.093	±6.53	±0.303	±10.88	±0.092	±9.93	±0.072	±7.92	±0.123
			(32.10)	(31.10)	(63.40)	(58.30)	(113.10)	(83.90)	(28.20)	(27.10)	(59.10)	(55.60)	(97.40)	(78.10)

TABLE 3

Alanine aminotransferase (AlAT) and aspartate aminotransferase (AAT) activity levels in selected tissues of Fish exposed to lethal (0.15 ppm) and sublethal (0.05 ppm) concentrations of lindane. Protein/h. Each value is $\bar{x}$ ISD of 6 observations. The values in parenthesis are per cent change over control. Values are expressed as μ moles of pyruvate formed/mg. protein/h. All values are significant at $P < 0.001$ except a : Not significant ; b : $P < 0.05$, c : $P \leq 0.025$; d : $P < 0.01$

Tissue	Control		Experimental											
			Lethal exposure h						Sublethal exposure h					
			12		24		48		12		24		48	
	AlAT	AAT	AlAT	AAT	AlAT	AAT	AlAT	AAT	AlAT	AAT	AlAT	AAT	AlAT	AAT
Brain	4.26	1.88 ±0.13	5.98^{b} ±0.18 (40.0)	0.70^{b} ±0.08 (-34.0)	6.05 ±0.22 (61.70)	1.92^{b} ±0.05 (+16)	9.69 ±0.62 (126.0)	2.42 ±0.04 (+34)	5.90^{d} ±0.23 (38.5)	0.55^{b} ±0.03 (-46)	6.63 ±0.22 (55.6)	1.61^{a} ±0.04 (-5)	8.51 ±0.74 (98.3)	1.98^{d} ±0.13 (+15)
Liver	4.64 ±0.78	1.54 ±0.10	6.13^{d} ±0 44 (27.6)	1.50^{a} ±0.11 (-3)	8.69^{b} ±0.49 (80.6)	1.69^{b} ±0.13 (+9)	10.37 ±0.66 (145.1)	2.54 ±0.12 (+64)	5.89^{c} ±0.19 (22.6)	1.34^{c} ±0.09 (-13)	7.28^{b} ±0.22 (51.8)	1.58^{a} ±0.12 (+4)	9.93 ±0.19 (106.8)	1.93^{d} ±0.15 (+25)
Muscle	1.94 ±0.14	1.67 ±0.22	2.14^{c} ±0.13 (41.4)	1.20^{d} ±0.07 (-40)	3.47^{d} ±0.11 (82.3)	1.96^{b} ±0.10 (+5)	4.63 ±0.38 (113.0)	2.92 ±0.13 (+75)	2.69^{b} ±0.13 (48.5)	1.13d ±0.05 (-42)	3.29^{b} ±0.14 (73.2)	1.70^{a} ±0.07 (+3)	3.72 ±0.70 (91.7)	2.24^{d} ±0.15 (+34)
Gill	1.34 ±0.09	1 49 ±0.26	1.58^{c} ±0.12 (17.8)	1.56^{a} ± 0.14 (+3)	1 66^{c} ±0.08 (23.3)	1.62^{b} ±0.13 (+7)	1.93^{d} ±0.10 (43.50)	1.93^{d} ±0.18 (+29)	1.46^{a} ±0.08 (8.8)	1.50^{a} ±0.08 (+1)	1.51^{a} ±0.11 (12.3)	1.59^{a} ±0.07 (+5)	1.63^{b} ±0.07 (21.2)	1.82^{b} ±0.20 (+22)

content form the structural moiety of a cell[10], it is likely that lindane interferes with the structural and movement sites of the muscular unit and also that it alters the contraction kinetics of muscle tissues as reported earlier in case of other OC compunds[13].

Corresponding with the decrease in the protein content, there is increment in free amino acid (TNPS) content and an increase in neutral protease (NP) activity. The order of tissue susceptibility to lindane toxicity with reference to TNPS and NP is liver, muscle, brain and gill. This clearly indicates that lindane induces proteolysis in the corresponding tissues inducing hepatotoxicity and possibly muscular atrophy. Moreover, an elevation in TNPS content in the tissues suggests a drop in tissue energy requirements and the tissues utilise TNPS content for energy production. The enhancement of AlAT has been more than AAT in the tissues. The increase in AAT and AlAT is known to indicate stress conditions[14].

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